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The Peripheral Resistance in Arteries of Legs Is Inversely Proportional to the Severity of Chronic Venous Insufficiency

KEN MALANIN[1], ARTO HAAPANEN[2], PERTTI J. KOLARI[1], INKERI HELANDER[1], and VÄINÖ K. HAVU[1]

Departments of [1]Dermatology and [2]Radiology, University of Turku, Turku, Finland

There are conflicting reports in the literature about the existence of arteriovenous shunting in legs with chronic venous insufficiency. Using duplex scanning, we have earlier shown that there is lowered peripheral resistance in the arteries of legs with venous ulcer together with premature venous filling in angiography.

In the present study we investigated the peripheral resistance in the arteries of 16 legs with chronic venous insufficiency without present ulcer. We compared the results with those obtained from 12 healthy legs and from 18 legs with venous ulcer.

There was a highly significant inverse correlation between the severity of chronic venous insufficiency and the peripheral resistance in the popliteal, the dorsal pedal and the posterior tibial arteries ($p < 0.001$).

These results suggest that there is arteriovenous shunting in legs with chronic venous insufficiency and that this phenomenon correlates with the degree of chronic venous insufficiency. ***Key words: arteriovenous shunting; duplex scanning.***

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K. Malanin, Tanhuantie 7 D, 53920 Lappeenranta, Finland.

The cutaneous blood flow is consistently found to be increased in legs with chronic venous insufficiency (CVI) (1–3), despite the fact that there are lowered transcutaneous skin oxygen tension (1, 2, 4) and predisposition to ulcer formation. Premature venous filling is a common finding in angiography of legs with CVI (5–7), and the oxygen content of venous blood in those legs is elevated (5, 8). It has been suggested that this phenomenon is due to arteriovenous shunting (av-shunting), present in legs with CVI (5–7).

The existence of av-shunting in legs with CVI has, however, been challenged (9–12) on the basis of labelled protein perfusion studies (9, 10).

Recently we could confirm the premature venous filling detected by angiography in legs with venous ulcer. Using duplex scanning we found significantly lowered peripheral resistance in the popliteal and the posterior tibial arteries in those legs compared with healthy legs (13).

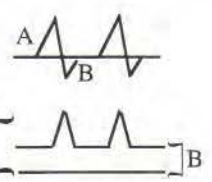
Because the pathogenesis of venous leg ulceration is not fully understood (14) and the existence of av-shunting has been challenged, further studies are needed to clarify these issues. The aim of the present study was to compare the peripheral resistance in arteries of legs with CVI without ulcer with healthy legs and with legs with venous ulcer.

PATIENTS AND METHODS

After informed consent had been obtained, 23 subjects (16 women, 7 men) were studied. The patients had no diabetes, peripheral neuropathy or symptoms of peripheral arterial disease. The legs were divided into three clinical groups (Class 0, Classes 1–5 and Class 6) according to the new classification system presented at the American Venous Forum's 6th Annual Meeting in 1994 (15). The clinical and anatomical classifications of the legs are presented in Table I.

The patients were examined in a quiet room with an ambient temperature of 23°C. The legs were without any compression bandaging or stocking at least from the evening before the day of investigation.

The arterial blood flow in the popliteal, dorsal pedal and posterior tibial arteries of the recumbent patient was measured using the duplex method with an Acuson 128 colour flow ultrasound scanner (Mountain view, Ca, USA). The recordings of these arteries were taken from the popliteal fossa, from the dorsum of the foot and from behind the medial malleolus, respectively. Doppler spectra of the arteries were recorded as hard copies. In order to obtain objective quantitative information about the peripheral resistance, resistance indices (RI) of arteries were calculated from Doppler waveforms as follows (16):



$$RI = \frac{A + B}{A} \qquad RI = \frac{A - B}{A}$$

RI is more than 1 when there is reverse flow component in early diastole and RI is 1 when there is no reverse flow. When there is antegrade flow during the whole diastole the RI is less than 1.

Table I. *The clinical and anatomical classification of the legs*

Clinical classification: Class 0: No visible or palpable signs of venous disease ($n = 12$), Class 1: Telangiectases or reticular veins ($n = 2$), Class 2: Varicose veins ($n = 2$), Class 3: Oedema ($n = 2$), Class 4: Skin changes ascribed to venous disease (e.g. pigmentation, venous eczema, lipodermatosclerosis) ($n = 7$), Class 5: Skin changes as defined above with healed ulceration ($n = 3$), Class 6: Skin changes as defined above with active ulceration ($n = 18$).

Anatomical classification: S = Superficial venous insufficiency, D = Deep venous insufficiency, P = Perforating vein insufficiency. One or more system(s) involved.

Anatomical classification	Clinical classification		
	Class 0	Classes 1–5	Class 6
S		3	
D		1	
P		4	2
SD		1	
SP		2	5
SDP		2	5
DP		3	6
Number of legs	12	16	18
Female/Male	6/6	11/5	15/3
Age, years mean, range	63.3 (51–72)	66.6 (53–86)	65.9 (51–86)

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These calculations were made from the hard copies on a separate occasion without knowledge of the clinical data.

Statistics

Results are expressed as median and ranges. Statistical testing was performed using Student's *t*-test for unpaired groups with unequal variances. Statistical significance was set at two-tail $p<0.05$. Non-significant results are expressed as ns.

The relationship between the classes of CVI and RI was analyzed using a linear regression model.

RESULTS

The resistance indices are expressed in Table II. Recordings could not be obtained from one dorsal pedal artery and from two posterior tibial arteries. The RI was significantly lowered in the dorsal pedal and the posterior tibial arteries in the legs of Classes 1–5 compared with healthy legs. The RI was further significantly lowered in the popliteal and the posterior tibial artery in the legs with venous ulcer (Class 6) compared with the legs of Classes 1–5.

In Fig. 1 is indicated the relationship between the CVI classes and the RI in the arteries separately and in all arteries. There is a highly significant inverse correlation between the severity of the CVI and the RI in all arteries separately and together ($p<0.001$).

DISCUSSION

Our results show that the peripheral resistance in the arteries of legs has a significant inverse correlation with the degree of venous stasis in leg. The lowest arterial resistance was in the legs with venous ulcer. There was a great variance in the RI-values in the legs with CVI, especially in the legs with present ulcer. These findings suggest av-shunting, which may be a fluctuating phenomenon, and are in concord with the following earlier findings in legs with CVI:

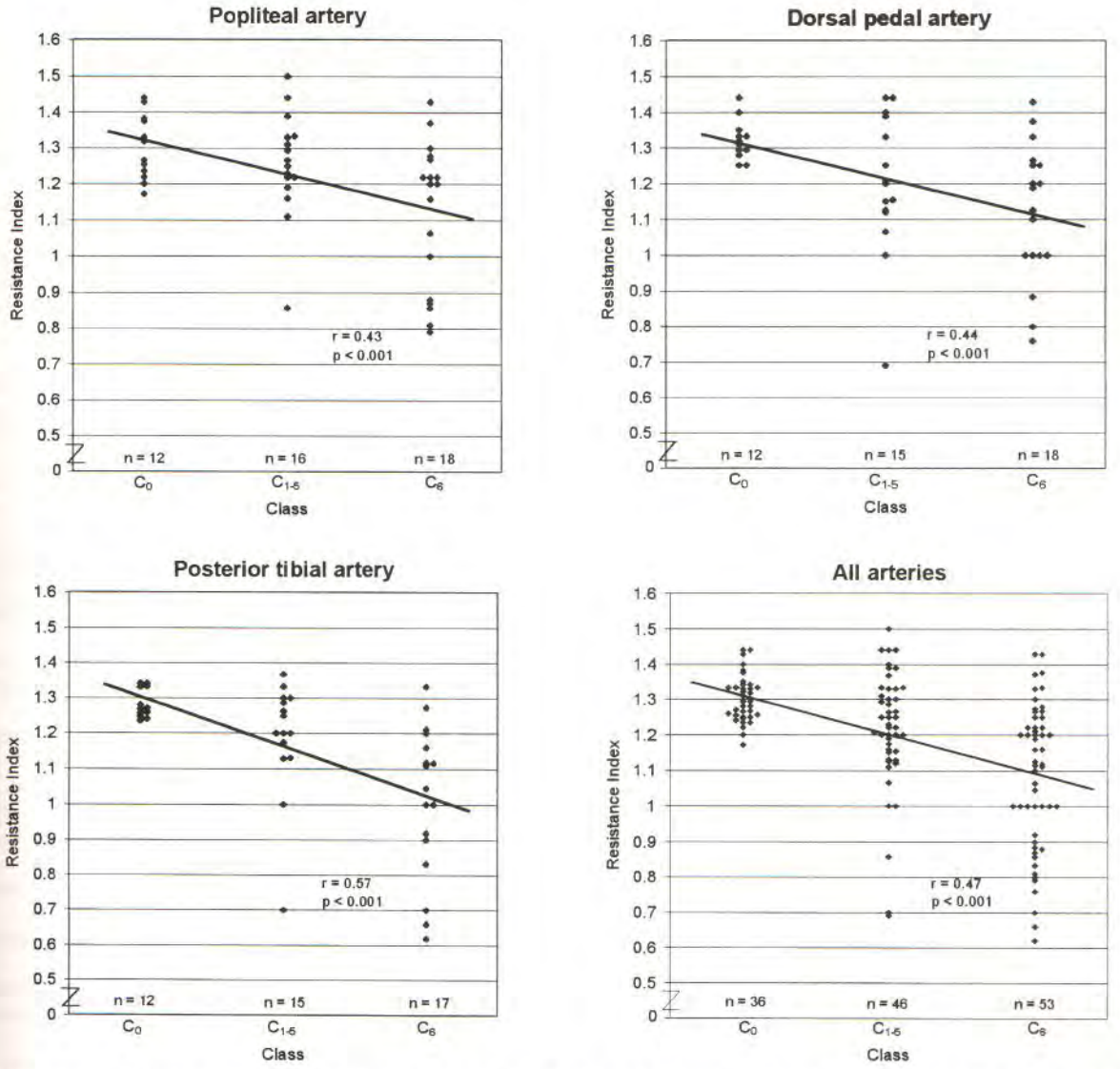

Fig. 1. The severity of chronic venous insufficiency in the legs vs. the peripheral resistance (resistance index) obtained from the popliteal, the dorsal pedal and the posterior tibial arteries. The relationship is shown by using a linear regression model. C_0 (Class 0): 12 healthy legs, C_{1-5} (Classes 1–5): 16 legs with chronic venous insufficiency without present ulcer, C_6 (Class 6): 18 legs with venous ulcer.

Table II. *The severity of chronic venous insufficiency in the legs and the peripheral resistance (resistance indices) obtained from the popliteal, the dorsal pedal and the posterior tibial arteries*

Class 0: 12 healthy legs, Classes 1–5: 16 legs with chronic venous insufficiency without present ulcer, Class 6: 18 legs with venous ulcer. Median and range values.

	Resistance index		
	Popliteal artery	Dorsal pedal artery	Posterior tibial artery
Class 0	1.29 (1.17–1.44) $n=12$	1.32 (1.25–1.44) $n=12$	1.27 (1.24–1.34) $n=12$
Classes 1–5	1.26 (0.86–1.50) $n=16$	a 1.20 (0.69–1.44) $n=15$	a 1.20 (0.70–1.37) $n=15$
Class 6	b 1.20 (0.79–1.43) $n=18$	1.16 (0.76–1.43) $n=18$	b 1.05 (0.62–1.33) $n=17$

a: $p<0.05$ compared with Class 0.
b: $p<0.05$ compared with Classes 1–5.

- The cutaneous blood flow is increased (1–3).
- The capillary density of the skin is diminished (17, 18).
- The transcutaneous oxygen tension ($TcPO_2$) is lowered (1, 2, 4).
- The oxygen extraction is reduced in the skin (19).
- The oxygen content of venous blood is elevated (5, 8).
- There is premature venous filling in angiography (5–7, 13).

The findings of increased capillary density of the skin in legs with CVI, observed in some histological investigations (3, 20), can be due to sections through elongated and tortuous capillaries, which is typical of the reduced capillary bed of skin in CVI (1, 17, 21). Thus, according to the above-mentioned findings (1–5, 8, 17–19) it is not believable that lowered peripheral resistance in arteries would be due to increased local capillary bed caused by e.g. chronic inflammation.

In the literature there are conflicting reports concerning the venoarteriolar response (VAR) in legs with CVI. In some reports it has been found to be impaired (3, 22) or better (23, 24) in legs with CVI compared to healthy legs. In our former investigation the VAR in legs with severe CVI was not impaired compared to healthy legs (25). Thus, it seems improbable that the lowered peripheral resistance in legs with CVI could be caused by impairment of the VAR.

Based on the findings of isotope studies, the existence of av-shunting has been challenged (9–12). Those studies have, however, been performed without correlation to angiography, and albumin may act like fibrinogen and depose pericapillarly in postthrombotic legs (12). This may explain why labelled albumin can accumulate near the ulcer (9). Thus, the results of radioisotope studies may be misleading in attempts to examine arteriovenous shunting in legs with CVI.

Postraumatic av-shunting is known to cause leg ulcers (26) and artificial av-anastomoses made on the arm for haemodialysis can cause digital ulcers with hyperpigmentation, induration and pericapillary fibrin formation, which all are typical of venous ulcer (27, 28). There is also evidence that diabetic microangiopathy can cause av-shunting, which may contribute to diabetic foot ulcers (29).

In conclusion, our results support the idea that chronic venous insufficiency may be coupled with arteriovenous shunting. Further studies are needed to clarify the significance of this phenomenon in the pathogenesis and healing of venous leg ulcers.

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